Delayed onset of neurogenic pulmonary oedema following an evolving ischaemic stroke

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SUMMARY

Any insult to the central nervous system can lead to the rare occurrence of neurogenic pulmonary oedema (NPO). It is usually associated with significant neurological injury (eg, subarachnoid haemorrhage or traumatic brain injury) with a relatively rapid onset. As an exception to this observation, we report a middle-aged woman who developed NPO 72 hours after the onset of a subtle but evolving right middle cerebral artery infarction confirmed on CT. Aggressive use of diuretics and vasodilators, as is normally the case for cardiogenic pulmonary oedema, can compromise cerebral blood flow and the ischaemic penumbra. This case illustrates how the diagnostic and therapeutic challenges were successfully addressed with the aid of bedside ultrasonography and close haemodynamic monitoring to reverse the respiratory failure while protecting the brain.

BACKGROUND

Neurogenic pulmonary oedema (NPO) is defined as an acute non-cardiogenic pulmonary oedema that usually develops within minutes to hours following a neurological insult. A delayed onset beyond 24 hours, as in our case, is not well recognised in the literature. Despite being first described in the 17th century, it remains a poorly understood clinical entity that is often mistaken as a presentation of left ventricular failure, pneumonia or sepsis with acute respiratory distress syndrome.[1 2] Misdiagnosis can be deleterious because unique to NPO, aggressive diuresis to control pulmonary volume overload or permissive hypercapnia during mechanical ventilation can potentially compromise cerebral perfusion and risk further neurological damage.[3] To highlight these challenges, we present a case of delayed onset NPO secondary to an evolving ischaemic stroke.

CASE PRESENTATION

A middle-aged lady with a background of hypertension, type 2 diabetes mellitus and essential thrombocythaemia presented with a 3-day history of progressively worsening slurred speech and recent onset left facial droop, which resolved within the first 4 hours of admission. Being right hand dominant, she denied any arm or leg weakness, sensory disturbances or swallowing difficulties. There was no history of postural headaches, vomiting, diplopia or fever. Over the preceding year, blood pressure (BP) was well controlled on amlodipine 10 mg and HbA1c levels maintained below 42 mmol/mol on sitagliptin 5 mg and glimepiride 2 mg, indicating good diabetic control. Essential thrombocythaemia had been managed with the platelet-reducing agent, anagrelide, achieving a satisfactory platelet count of 530×10^9/L with stable leucocytosis. She does not smoke or consume alcohol, and after retiring from nursing, keeps active with daily exercises. On examination, she was apyrexic with a regular heart rate (HR) of 90 beats per minute, BP 130/80 mm Hg and oxygen saturation 97% on room air. There was subtle dysarthria with no evidence of facial paresis. No other localising neurological deficit was found. She appeared comfortable with normal vesicular breath sounds and no heart murmurs on auscultation or peripheral oedema.

The suspicion of ischaemic stroke was confirmed on CT head. This revealed an evolving infarct in the right middle cerebral artery territory and excluded intracranial haemorrhage or space-occupying lesion (figure 1). Her National Institutes of Health Stroke Scale score (NIHSS) was 2. As she did not meet the criteria for thrombolysis, she was given 300 mg aspirin orally and transferred to the acute medical unit.

Six hours postpresentation, she suddenly deteriorated. Desaturating to 80% on 15 L/min non-rebreather mask, she exhibited central cyanosis and bilateral coarse inspiratory crackles with monophonic expiratory wheeze, consistent with pulmonary oedema. Jugular venous pressure did not appear raised. BP was 226/60 mm Hg and HR 85 beats per minute. An ECG showed sinus rhythm with first-degree atrioventricular block and premature atrial complexes, but no signs of an acute or old myocardial infarction (figure 2). No intravenous fluids were given beforehand to have caused iatrogenic fluid overload.

INVESTIGATIONS

She became drowsy (Glasgow Coma Scale (GCS) 14/15 for confusion) and developed acute type 2 respiratory failure with mixed acidosis on arterial blood gas (pH 7.026, $Paco_2$ 14.1 kPa, Pao_2 8.7 kPA, HCO_3 16.5 mmol/L, base excess −3.6 mmol/L, lactate 3.3 mmol/L). Pao_2:Fio_2 65 mm Hg suggested severe hypoxia. A central cause for the respiratory failure was unlikely since there was no new neurological deficit and a repeat CT head excluded haemorrhagic transformation or worsening infarct. This in turn made aspiration pneumonia unlikely, supported further by the initial normal swallow and ruled out on chest radiography (CXR). Instead, CXR revealed bilateral pulmonary infiltrates consistent with acute pulmonary oedema (figure 2).

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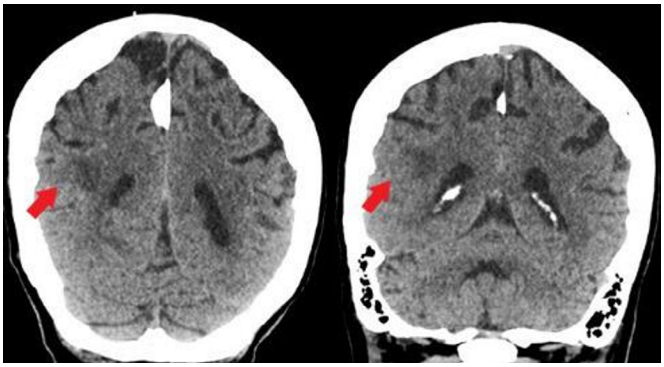


Figure 1 Non-contrast CT head in sagittal and coronal planes demonstrating subtle loss of the grey-white differentiation within the posterior right frontal lobe (see arrows), indicating an evolving infarct in the right middle cerebral artery territory without evidence of intracranial haemorrhage or space-occupying lesion.

DIFFERENTIAL DIAGNOSIS

It was crucial to differentiate between cardiogenic versus non-cardiogenic pulmonary oedema early on since this not only influenced immediate management but also place of care in the context of an acute stroke. First, sepsis-related acute respiratory distress syndrome was ruled out. She displayed no signs of infection clinically and biochemically (C reactive protein 4 mg/L and White cell count (WCC) 18 × 10^9/L, which had been unchanged for years due to her underlying myeloproliferative disease). COVID-19 PCR was negative.

Cardiogenic pulmonary oedema was the primary differential to consider. There was no history or clinical features of chronic heart failure (HF). Admission N-terminal-prohormone brain natriuretic peptide (NT-proBNP) was 399 pmol/L, but as highlighted by National Institute for Health and Care Excellence (NICE) guidance on HF, BNP can be acutely elevated by hypoxia, elevated pulmonary capillary hydrostatic pressure and tachycardia from sympathetic hyperactivity.[4] Repeat ECG was unremarkable. Serial troponins (34 and 32 ng/L) excluded an acute myocardial infarction. An urgent bedside echocardiography was performed by a senior cardiac physiologist (video 1). Left ventricle was non-dilated with normal systolic function (ejection fraction >60%). Diastolic function was normal with non-dilated atria. Mitral inflow Doppler confirmed sinus rhythm. There were no signs of valvular dysfunction or left ventricular thrombus. Cardiologist opinion was in favour of a non-cardiogenic cause of pulmonary oedema.

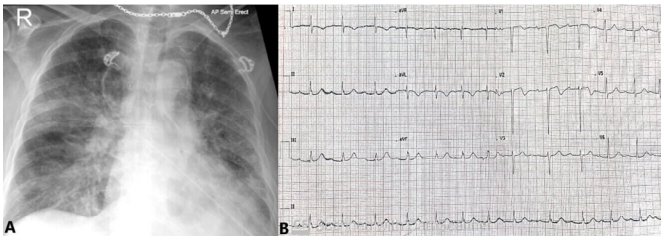
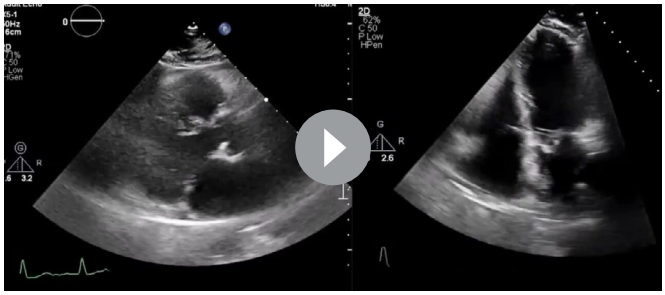


Figure 2 (A) Antero-posterior chest radiograph showing bilateral perihilar pulmonary infiltrates with Kerley B lines, peribronchiolar cuffing and cephalisation consistent with pulmonary oedema. (B) Admission ECG showing sinus rhythm with premature atrial complexes but no evidence of ischaemic changes or previous myocardial infarction. As per old ECGs, fixed biphasic T waves in V1–2 are present which was considered as a physiological variant for the patient.



Video 1 Parasternal long-axis and apical four-chamber view showing normal left ventricular systolic function with no regional wall motion abnormalities to suggest myocardial infarction.

Other causes of flash pulmonary oedema include renal artery stenosis (RAS). This patient has a history of chronic kidney disease stage 3 (diabetic nephropathy) with estimated glomerular filtration rate 35 mL/min/1.73 m^2 and microalbuminuria 19 mg/mmol, making RAS a possible cause. However, she had a normal renal ultrasound few months prior to admission and a repeat inpatient ultrasound demonstrated normal bilateral renal lengths (10–11 cm) with renal arterial Doppler profile. Furthermore, there were no refractory hypertension, abdominal bruit or hypokalaemia. Another cause is hypertensive crisis. Although this shares a common underlying mechanism with NPO in terms of adrenergic overactivity, she did not present with accelerated hypertension. Her BP had been well controlled on amlodipine alone over the last 12 months. Finally, although she was not thrombolysed, an interesting differential includes fibrinolytic-related pulmonary oedema. According to the British National Formulary, this is classed as a common side effect of fibrinolytic agents (eg, alteplase).

As a diagnosis of exclusion, she was managed as NPO due to the evolving stroke. No atrial fibrillation on continuous cardiac monitoring or mural thrombus on echocardiography was found to suggest an embolic stroke, and the thrombocythaemia had been unchanged, making atherosclerosis the most probable cause of stroke. Stroke-in-evolution can be aggravated by metabolic derangements, haemorrhagic transformation and hypoxia, all of which were excluded on admission.[5]

TREATMENT

With the onset of acute pulmonary oedema, it was vital to address this urgently with intravenous diuretics and glyceryl trinitrate (GTN) infusion since hypoxia worsens ischaemic stroke. However, given the presence of an evolving stroke, there was a concern of compromising cerebral perfusion and the ischaemic penumbra, should arterial BP fall too quickly with vasodilators. Our local hospital guidance on GTN infusion advises an initial rate of 1.2 mg/hour for acute pulmonary oedema, but considering the above, it was initiated at 0.5 mg/hour. A stat dose of intravenous furosemide 50 mg was given, followed by close BP monitoring. Bedside echocardiography helped to assess volume status. This showed a collapsible non-dilated inferior vena cava, suggesting a normal right atrial pressure between 0 and 5 mm Hg. Estimated stroke volume was normal and estimated left ventricular filling pressures were not raised (figure 3). No further intravenous diuretics were required. GTN infusion was continued at 0.5 mg/hour for the following 6 hours, maintaining the systolic BP between 160 and 180 mm Hg. At this point, her clinical condition improved with a dramatic reduction in oxygen requirements and resolution of type 2 respiratory failure (Arterial blood gas (ABG) on 2 L/min O_2: pH 7.42, PO_2 9 kPa, PCO_2

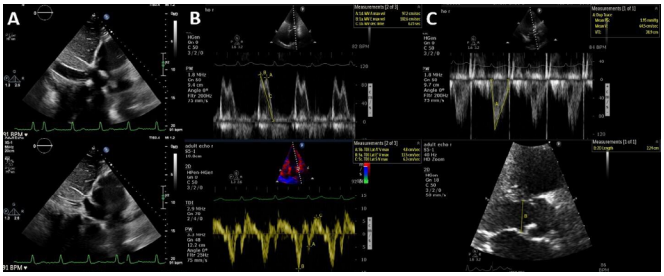


Figure 3 Estimation of haemodynamics using bedside echocardiography. (A) Inferior vena cava was <2.1 cm and fully collapsible on inspiration, estimating right atrial pressure 0–5 mm Hg; (B) Ratio of early diastolic mitral inflow E max to mitral annular e' velocity estimates left ventricular filling pressure. Here it is 7.6 (<13 normal); (C) Estimation of stroke volume (82 mL) and cardiac output (7 L/min) based on heart rate, diameter and forward flow Doppler of the left ventricular outflow tract.

5.23 kPa, HCO_3 25 mmol/L, BE 1.4 mmol/L, P/F ratio 242 mm Hg).

OUTCOME AND FOLLOW-UP

Over the following 12 hours, her breathlessness fully resolved and she was successfully weaned off oxygen. No further cardiorespiratory deterioration occurred during the admission. The dysarthria was still present but no other neurological deficit had developed; NIHSS was 1. An MRI with diffusion-weighted imaging (DWI) was not performed in this case. The multidisciplinary stroke team felt that it was prudent to avoid an extended period of lying flat in the MRI scanner, given the recent resolution of pulmonary oedema. Furthermore, despite its higher diagnostic sensitivity in detecting acute ischaemia, it was felt that further imaging would unlikely influence the management and prognosis in light of the improving clinical status, low NIHSS at day 4 and the low likelihood of stroke mimics (eg, neoplastic or infectious processes). There was no concern from the haematology team with regards to the essential thrombocythaemia in the setting of an acute stroke. They advised the patient to remain on the same dose of anagrelide on top of the 2-week course of oral aspirin 300 mg for acute stroke. After receiving stroke rehabilitation, she was discharged. Over the following 6 weeks, she has not had further episodes of pulmonary oedema or reports of breathlessness.

DISCUSSION

Any insult to the central nervous system (CNS) can lead to the rare occurrence of NPO which is associated with high morbidity and mortality if not recognised or treated promptly.[6] This is hindered by the lack of diagnostic indicators and universal diagnostic criteria. Its pathophysiology remains unclear. The most well-reported mechanisms include: (1) an elevated sympathetic nervous activation with catecholamine surge, leading to systemic and pulmonary vasoconstriction and elevated capillary hydrostatic pressure; (2) damage of the blood-brain barrier with resultant secretion of inflammatory factors, causing alveolar wall injury and increased pulmonary vascular permeability.[7] Both pathways eventually cause alveolar and interstitial pulmonary oedema.[8]

Severe traumatic brain injury, intracranial haemorrhage and seizures are the commonly reported neurological insults associated with NPO (table 1).[9–17] On the other hand, ischaemic stroke is a relatively rare trigger. The provoking infarction is usually large and clinically significant, often requiring intravenous thrombolysis or neurosurgical intervention.[18–20] In this case, the stroke was subtle with a low NIHSS. Interestingly, the right insular cortex mainly controls sympathetic nervous activity and so, NPO is closely related to right insular infarction.[21] This may explain why our patient developed NPO over time as the evolving ischaemia in the right middle cerebral artery territory also have affected the right insular cortex (figure 1).

NPO usually arises rapidly within the first few hours of presentation, particularly in the setting of traumatic brain injury and acute intracranial haemorrhage, as highlighted in table 1.[3 11 12 14] We found one case report of delayed NPO occurring 4 days after the onset of subarachnoid haemorrhage; however, at that time, there was angiographic evidence of significant cerebral arterial spasm which could have triggered the NPO.[10] Another case highlighted a delayed NPO at day 7 of admission for traumatic brain injury; however, this occurred on the day of a lumbar puncture (for cistern drainage due to raised intracranial pressures) which could have induced another brain injury.[13] With regards to acute stroke, the time of onset is usually rapid (table 1). Raja *et al* described a case of NPO that developed an hour after the occurrence of a lateral medullary infarct secondary to a vertebral artery dissection.[19] A review by Zhao *et al* found that the majority of NPO cases post stroke occur within 4 hours and in rare instances, up to 2 days post presentation of stroke.[3]

Table 1 Summary of case reports of neurogenic pulmonary oedema in terms of neurological injury, time of onset and the absence or presence of myocardial dysfunction

Cases	Potential trigger	Onset of NPO from time of cerebral insult	BNP levels	Echo
Simmons *et al*[9]	Head injury (gunshot)	Within minutes	Not reported	Not available; autopsy
Fisher *et al*[10]	Subarachnoid haemorrhage	After 4 days	Not reported	Normal
Loughnan *et al*[11]	Traumatic brain injury	Within minutes	Not reported	Not reported
Kerr *et al*[12]	Head injury	Within minutes	Not reported	Severe LVSD
Park *et al*[18]	Left middle cerebral artery infarction	Not specified; likely few hours	'Normal'	Normal
Lin *et al*[13]	Traumatic brain injury	At day 7 of admission	993 pmol/L	LVEF 50%
Raha *et al*[14]	Acute subdural haematoma	Within 30 min	482 pmol/L	Not reported
Raja *et al*[19]	Acute lateral medullary stroke	Within 1 hour of admission	Not reported	Not reported
Aljishi *et al*[20]	Vertebral artery dissection	Within 3 hours of admission	358 pmol/L	Normal
Sacher *et al*[15]	Epilepsy with tonic-clonic seizures	Within minutes	'Normal limits'	Normal
Yasumoto *et al*[16]	Epilepsy with tonic-clonic seizures	Within minutes of seizure	Not reported	Normal
Lo-Cao *et al*[17]	Severe traumatic brain injury	Within minutes	Not reported	Normal

BNP, brain natriuretic peptide; LVEF, left ventricular ejection fraction; LVSD, left ventricular systolic dysfunction; NPO, neurogenic pulmonary oedema.

Our case highlights that the time of onset of NPO can be delayed by up to 72 hours. This may be explained by the relatively consistent pattern of infarct growth over time as demonstrated in previous studies.[22 23] For example, Lansberg *et al* found that on diffusion-weighted MRI of untreated stroke patients (ie, not thrombolysed) such as in our case, the early DWI lesion size reached a maximum volume at mean 74 hours, followed by a subsequent reduction in size from day 4 to 7.[23] Hence in our case, it may be postulated that the NPO occurred once the insular cortex was involved by the expanding lesion on day 3, followed by subsequent improvement in clinical status from day 4 onwards. It has been suggested that the early expansion of the DWI lesion size on day 1 reflects the infarct core (due to restricted diffusion of intracellular water), while the subsequent growth on days 2–3 reflects the formation of vasogenic oedema.[23 24] Although not currently part of routine clinical practice, the use of perfusion-weighted imaging (PWI) to evaluate the DWI-PWI mismatch (ie, differentiation of the infarct core from the hypoperfused ischaemic penumbra) may better demonstrate this progression of an evolving ischaemic stroke.[24]

Apart from the challenging diagnosis, managing such patients can be difficult. This is largely conservative by addressing the underlying CNS injury, controlling circulatory overload and correcting haemodynamic derangements.[2 3 13 17 25] However, this becomes even more challenging when faced with acute haemodynamic changes in the presence of evolving stroke. On the one hand, administering vasodilator drugs and intravenous diuretics are crucial to reduce the pulmonary volume overload. On the other hand, avoiding a sudden reduction of BP and consequential cerebral hypoperfusion is of paramount importance to prevent jeopardising CNS recovery. Our case demonstrates a volume-based treatment approach guided by fluid status assessment and echocardiography. Bearing in mind the limitations of echocardiography in assessing volume status, it remains a valuable tool in detecting significant hypovolaemia or hypervolaemia.[26] A dilated inferior vena cava with <50% inspiratory collapse suggests a raised right atrial pressure. Evidence of volume overload would be further supported by evidence of raised left ventricular end-diastolic pressure as reflected by an elevated E/e' > 13 (figure 3).

It was also vital to rule out left ventricular systolic dysfunction (LVSD). This would have warranted cardiac monitoring in a high-dependency setting. NT-proBNP is a good initial screening test for LVSD but as mentioned above, it is not highly specific. NT-proBNP levels in the setting of NPO has been reported either as normal, moderately raised between 350 and 480 pmol/L similar to our case or above 900 pmol/L (table 1).[13 14 20] Indeed, Takotsubo cardiomyopathy (ie, stress-induced or broken-heart syndrome) can also occur concurrently with NPO since it also shares the pathophysiology of sympathetic overactivation and catecholamine surge, which can cause direct myocardial toxicity. This was illustrated in a case following right insular cortex infarction.[27] Another case described the occurrence of NPO immediately after the clamping of the carotid artery during carotid endarterectomy, after which the patient was also found to have Takotsubo cardiomyopathy on echocardiography; hence, as a caveat, NPO may rarely coexist with LVSD.[28] Above all, this case informs clinicians of another urgent indication for echocardiography (even out-of-hours) as it clearly changes patient management and place of care.

Patient's perspective

Experiencing sudden shortness of breath was very frightening. I was petrified. I hope the manuscript will raise awareness and inform doctors about this emergency in order to help in diagnosis and management of other people with a similar presentation. I will always be grateful to the doctors who worked hard and saved my life that night.

Learning points

- Neurogenic pulmonary oedema can occur secondary to any neurological insult regardless of severity such as in this case of an evolving stroke with relatively mild symptoms and signs.
- In contrast to the classical acute presentation (ie, few hours), it can also present in a delayed fashion after several days after neurological injury.
- Prompt recognition is vital to prevent aggressive diuresis and vasodilator use in order to avoid compromising cerebral perfusion and worsening of any salvageable ischaemic penumbra.
- Management can be guided by the use of point-of-care focused echocardiography to estimate cardiac output and volume status, supporting its value in the acute medical setting.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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